

**Topic:** Mutism and Amnesia Following High-Voltage Electrical Injury: Psychogenic Symptomatology Triggered By Organic Dysfunction?

**Authors:** Dr. Nishant K. Mishra, MBBS (1, 2), Dr. Heike Russmann, MD (1), Dr. Cristina Granziera, MD PhD (1), Prof. Philippe Maeder, MD (4), Prof. Jean-Marie Annoni, MD (1,3).

**Affiliations:**

1. Department of Neurology, University Hospital of Lausanne (CHUV), Lausanne, Switzerland.
2. Acute Stroke Unit, Western Infirmary and Faculty of Medicine, Division of Cardiovascular and Medical Sciences, Church Street, Glasgow, G11 6NT, UK.
3. Neurology Unit, Department of Medicine, University of Fribourg and Fribourg County Hospital , Fribourg, Switzerland
4. Department of Radiology, University Hospitals of Lausanne, CHUV, Lausanne, Switzerland.

*Abstract: 238 words., main text 2183; 1 figure, 2 tables; Additional data*

,

**Corresponding Author:**

Dr. Nishant K. Mishra, Acute Stroke Unit, Western Infirmary and Faculty of Medicine, Division of Cardiovascular and Medical Sciences, Church Street, Glasgow, G11 6NT, Scotland. Email: nishmishra@gmail.com Tel: +44-7596006875

**Abstract**

**Background:** Mutism and dense retrograde amnesia are found both in organic and dissociative contexts. Moreover, dissociative symptoms may be modulated by right pre-frontal activity. A single case, MR, developed left hemiparesis, mutism and retrograde amnesia after a high-voltage electrocution without evidence of lasting brain lesions. MR suddenly recovered from his mutism following a mild brain trauma two years later.

**Methods:** MR's neuropsychological pattern and anatomoclinical correlations were studied through *i)* language and memory assessment to characterize his deficits, *ii)* functional neuroimaging during a standard language paradigm, and *iii)* assessment of frontal and left insular connectivity through Diffusion Tractography Imaging and Transcranial Magnetic Stimulation. A control evaluation was repeated after recovery.

**Findings:** MR recovered from the left hemiparesis within 90 days of the accident, which indicated a transient right brain impairment. One year later, neurobehavioral, language and memory evaluations strongly suggested a dissociative component in the mutism and retrograde amnesia. Investigations (including MRI, fMRI, diffusion tensor imaging, EEG and r-TMS) were normal.

Twenty-seven months after the electrical injury, MR had a very mild head injury which was followed by a rapid recovery of speech. However, the retrograde amnesia persisted.

**Discussion:** This case indicates an interaction of both organic and dissociative mechanisms in order to explain the patient's symptoms. The study also illustrates dissociation in the time course of the two different dissociative symptoms in the same patient.

**Keywords:** Mutism, Dissociative disorders, Electrocution, Amnesia

### **Introduction:**

Mutism is characterized by the inability of a patient to produce any oral utterance despite intact vigilance and other language abilities<sup>1</sup>. It may occur in isolation or in association with akinesia following specific lesions in the cingulum, the supplementary motor area and mesencephalon, left or right frontal lobe lesions, callosotomy, parasagittal excisions and posterior fossa tumor resections, or in the frame psychogenic (conversion or dissociative) disorders<sup>1-6</sup>. Mutism constitutes 4.5% of all conversion symptoms, i.e., loss or distortion of neurological functions that cannot be fully explained by a known organic neurological disease (American Psychiatric Association, 1994)<sup>7</sup>. In this case, the symptom is isolated and generally has two variants: selective mutism and total aphonia<sup>8, 9</sup>. However, despite the fact that dissociative mutism may occur in association with a variety of psychosocial stressors, everyday clinical observations suggest that an organic brain dysfunction can play a triggering role<sup>10, 11</sup>. Dissociative amnesia is characterized by impaired retrieval processes of stored information, associated with the preserved ability to acquire new information. In addition, patients with this condition may display a state of “belle indifférence” towards their own situation and may manifest deficits in emotional processing. Interestingly, conversion disorders in different domains (motor domain, language, memory) have also been shown to be associated with focal brain functional impairment, mainly of the right frontal networks<sup>11-15</sup>.

The present study investigates a patient showing a left hemiparesis, mutism and a ten years' retrograde amnesia following a high-voltage electrocution. Electrocution and the associated physical trauma are known to cause neurological symptoms, e.g., due to brain lesions resulting from transient vascular instability, or clot disruption and embolization<sup>16-20</sup>. However, the patient recovered rapidly from his hemiparesis, but his mutism and his retrograde lacunar amnesia remained. In this study, detailed neurobehavioral and imaging investigation of the patient are reported which define the dissociative nature of the symptoms, despite the initial organic brain dysfunction. We also report the patient's recovery from mutism but not from amnesia, a finding

characterised by dissociation in the time course of the two different dissociative symptoms in the same patient.

### **Case Description and Methods:**

**Clinical Presentation:** MR, a right-handed, 40 year-old French male who worked as a sanitary installer, suffered a high-voltage electrical injury (7 x 400 volts) on September 17<sup>th</sup>, 2006 while repairing his electrical water heater. His family found him lying unconscious. On arrival at hospital, he was in a coma. After three hours he started to improve gradually, although he remained stuporose. He had a left-sided proportional hemiparesis (3/5), and electrical burns on his right index finger and the left side of his forehead were noted. He showed general improvement over the next two and a half days, from being stuporous to becoming fully alert and conscious. But he did not utter a single word. On the third day, he turned to his wife, made a sign that he wanted to write, and then began enquiring, by means of writing, about what had happened, where he was, etc. From the fourth day onwards, he could understand everything, including long sentences, but could communicate only by writing; he wrote long, meaningful texts without any difficulty. During these initial communications, the patient and his wife realized that he had no memories covering the past ten years. He believed that it was October 1996, and that he was recovering from a road traffic accident (MR had indeed suffered an accident with a mild head injury in October 1996). Medical evaluation confirmed mutism, left hemiparesis, and an intense amnesia covering all personal and world events during the period from October 1996 to September 17<sup>th</sup>, 2006. For example, he could not remember what he had done during this period, where he lived, what he did for a living or his family life. He had no memories of the political or sport events that had happened in the previous ten years, no knowledge of the attacks on the Twin Towers in New York on 9/11, no recognition of modern cell phones or computers (as these were too small and compact compared to the big mobile phones and heavy computers of ten years ago), etc. He was astonished by all the “new” objects that he discovered. Two CT scans

taken during the first week, and an MRI and EEG conducted after two weeks, were completely normal. The clinical follow-up is detailed in the Supplementary Materials section (Supplementary Material, Appendix 1), but he had more or less recovered from his hemiparesis in three months, while the mutism and lacunar amnesia for the period from 1996 to 2006 persisted for more than two years.

One year after his injury, he attended our Neurobehavioural Disorder Clinic at the request of his psychiatrist. At his first clinical evaluation, he displayed mutism, retrograde amnesia [personal episodic and semantic memory loss for the ten years prior to the accident, except for what he had relearned], anxiety, bucco-linguo-facial apraxia, and severe fatigue. We noted a reduced eye contact. His non-verbal language functions were clinically intact. On the IOWA scale for Personality Changes, his wife underlined the following behavioral modifications: irritability, depression, difficulty in planning, and decreased empathy towards other people. However, he showed emotional indifference to the accident; he explained in writing how he had coped with his impairment, and that he was not greatly bothered by his mutism. The psychiatric evaluation was normal and suggested mild depression.

### **Evaluation Methods**

In order to determine the phenomenology of the deficit, MR underwent a second Standard 3 Tesla MRI and a Brain Single Proton Emission Computerized Tomography (both normal). We undertook a detailed neuropsychological assessment (See Appendix-Table in the Supplementary Materials), but focused in greater detail on oral and written language as well as episodic and semantic memory. His language evaluation was conducted using the Montreal Toulouse Protocol [MT 86]. Memory performances were evaluated through classical neuropsychological tests (Span, Learning through Rey's Auditory-Verbal Learning Test, and Rey's Complex Figure) and the Kopelman's Autobiographical Memory Interview<sup>22</sup>. The Functional MRI involved two tasks presented in a block design: silent naming of the visually

presented stimuli and a word listening task (for more details, look in the experimental section). Standard motor evoked potentials and tractography were also achieved.

## Results

### *Language assessment*

The patient could write and communicate in writing. At times, he would just whistle. We failed to notice any articulatory/labial movement associated with phonation. There was an opening of the mouth and a correct “aaah” during the speech examination. But his mouth movements were incorrect when asked to repeat a sound (e.g., an attempt to repeat an “A” led to a complete closure of the lips). Laryngeal endoscopy showed normal movements of the vocal chord. We failed to find any other neurological symptoms. The Montreal Toulouse Protocol [MT 86] showed normal language performances except for his impossibility to vocalize: naming, repetition of words and non-words and oral reading were impossible (see Table 1).

### *Memory assessment*

His retrograde memory impairment was still dense, and recovery was patchy, and only partly followed Ribot’s Law<sup>21</sup>. For example, he correctly remembered the birth of his three daughters in 1991, 1992 and 1995, but he could not describe their childhood, schooling or holidays from 1996 to 2006. He could only describe the facts that he had relearned by talking to his family, or through what was described to him by his ex-colleagues and ex-boss. His general knowledge was intact, except for a significant number of social and political events that had happened during the period 1996-2006. Concerning the Twin Tower event, he explained that he had heard about the episode, but had no personal recollection of it. He had relearned how to use the new technical systems.

Standard Memory performances were normal, except for a slight decrease in short term memory (Table 2). Upon examination using Kopelman’s Autobiographical Memory Interview<sup>22</sup>, his scores were maximal for childhood and early adult life, and zero for recent episodes that had occurred during 1996-2006 (Table 2) that he had not

relearned through family and friends. He scored highly once more for the period between September 2006 and October 2007. On the Memory Malinger (TOMM) Test<sup>23</sup>, MR showed no evidence of malingering.

*Neuroimaging (fMRI Experiment):* Functional MRI involved two tasks presented in a block design: silent naming of the visually presented stimuli (man-made tools and animals) wherein the baseline was an attempt to produce non-verbal sounds; and word listening and recognition, wherein the baseline conditions were routine auditory nonverbal scenes (marketplace, street, beach, shop and railway station), each of which was presented six times. The environmental sounds lasted 2 s and started 1.5 s after the background onset<sup>24</sup>. Subjects were required to press a pneumatic device with their right hand in response to animal cries. In the naming task, we observed normal activity in Broca's area, the base of the central sulcus that projects over the facial motor area and the accessory para-median frontal motor area. In the verbal comprehension versus environmental sounds task, a stronger activation occurred in the left superior temporal gyrus (without any activation in Broca's area) and a bilateral activation in the planum temporal (Fig 1). These were reported to be normal, except for an unusual hyperactive focus in the left cerebellar hemisphere.

#### *Other Experimental sessions*

**DTI tractography** of the arcuate fasciculus bilaterally using a streamline reconstruction of the fibre trajectories as supported by Trackvis software was reported to be normal [See Supplementary Material, Appendix 2 and fig. 1d]. **Transcranial Magnetic Stimulation** showed normal central conductance, arguing against any long-term consequences of the electrocution to the central motor and premotor system (See online supplement).

**Follow up:** Two years and three months after the electrocution, MR suffered a fall after a Christmas party which resulted in a minor head injury and a transient ten

seconds' confusion. He then uttered a familiar French word, "merde" (shit), that he pronounced perfectly and which was distinctly heard by his colleagues, and began immediately to speak again. His speech was almost perfect in terms of articulation, but was characterized by hesitancy and short sentences of only four to five words that were used in combination with sign language. Although he could speak clearly, he remained slow in expressing himself during the first evening. Normalization of spontaneous speech took one week to occur.

On examination three weeks later he spoke normally, although the retrograde amnesia of the 1996-2006 period persisted. His wife noticed a change in his behavior (e.g., he had become hyperactive). Language evaluation was totally normal (Table 1). Memory assessment showed a slight impairment in learning, and the partial persistence of episodic and semantic amnesia for the period 1996-2006 in the Autobiographical Memory Interview<sup>22</sup> (Table 2). Follow-up neuropsychological evaluation pointed to mild difficulties in the performance of executive tasks (see table in the Additional Material). This mild residual cognitive impairment did not impair his daily and professional life. Structural MRI was still normal. A new experimental language session with Functional MRI, identical to the one described above, showed the same activations.

MR changed jobs once more and became a manager in an institute for deaf people, a position which he still holds two years later.

## **Discussion**

To the best of our knowledge, this is the first report of a patient developing mutism and dissociative amnesia following a high-voltage electrical injury. A particular feature of this clinical picture is that it has both "organic" and "dissociative" features. The baseline clinical features of our patient (i.e., coma followed by hemiparesis, mutism and retrograde amnesia) and the recovery of hemiparesis three months later strongly suggest a right brain dysfunction,<sup>1</sup> although none of the subsequent standard and experimental investigations (MRI, SPECT, EEG, DTI, f-MRI, TMS) showed any persistent abnormal findings. In the absence of a brain lesion or altered central



conduction, we hypothesise that initial transient alterations in neurons may have occurred from the electrical current, altering the electrical characteristics of cell membranes through mechanisms such as electroporation. Retrograde amnesia has been described in patients undergoing electroconvulsive therapy (ECT), even in the absence of demonstrated organic brain lesions<sup>25-28</sup>. It is quite common that cognitive problems which are noticed after the ECT are of short duration, and last only a few weeks.

One year later, the clinical pattern of mutism (total aphonia) fulfilled the criteria of a dissociative mutism as proposed by Akhtar and Blackman<sup>29</sup>: 1) sudden onset (3<sup>rd</sup> day after electrocution); 2) recent stressful event; 3) absence of long-term demonstrated organic disease (through neuroimaging); 4) selective lack of concern (“la belle indifférence”) about his mutism; and 5) symbolic meaning, in the sense that he started to work in an institution for deaf persons. Moreover, the rapid recovery two years later that was unrelated to therapy and the presence of oral apraxia but the partial ability to whistle, strongly suggest a functional component. Some closely-related classical organic syndromes, such as aphemia or opercular syndrome, were considered, but not retained; there was no dysarthria, vocalization ability (i.e., the ability for example to produce “aaaa”), which is generally maintained in opercular syndrome, was absent, as well as anarthria, drooling, and facial or masticator diplegia. Finally, normal pattern of brain activation during a language paradigm and unexpected sudden recovery are also strongly suggestive of dissociative phenomenology.

Concerning the amnesia, the pattern of retrograde massive amnesia fulfilled the criteria previously described in the context of dissociative amnesias (impaired retrieval process of stored information, while the acquisition of new information is conserved)<sup>33</sup>. The density of the amnesia, the parallel impairment of semantic knowledge which relates only to the period in question, the absence of a clear Ribot’s Law and the absence of learning impairment are also described in dissociative amnesia<sup>10</sup>. Such psychogenic amnesia are associated with various patterns of recovery<sup>10</sup>.

To summarise, we attribute a dissociative pattern to MR's symptoms, but which have been triggered by an initial transient organic brain dysfunction<sup>11</sup>. We suggest that an electrical current disrupted some specific language memory and memory networks<sup>18-20, 30, 34</sup>. The initial right frontal dysfunction is a putative trigger for both organic and psychogenic mechanisms<sup>31</sup>. This case also highlights some clinical evidence which indicates the interaction of an initial organic brain dysfunction and a consecutive psychogenic reaction<sup>11</sup>. Moreover, the simultaneous existence of two different dissociative symptoms (mutism and amnesia) in the same patient has so far not been described, although the multiplicity of dissociative disorders is a well-known phenomenon<sup>32</sup>. The recovery of speech and preservation of learning despite the persistence of a ten-year retrograde amnesia is worth reporting. This pattern of recovery is a further demonstration that, as in organic dysfunction, recovery from dissociative disorders can be selective. Additional study would be required to explore further the interaction of an initial transient organic brain dysfunction with a psychogenic response as exhibited by our patient.

**Acknowledgement:** Dr. N.K. Mishra was supported by the Swiss Federal Commission [ESKAS], Bern at the Department of Neurology, University Hospital of Lausanne [CHUV], Switzerland (2006-2008), and Prof. J-M. Annoni by the Swiss National Science Foundation, grants 320030-125196. The authors thank Dr. Niall Macdougall and Ann Travis for their comments and editing of the draft manuscript.

**Conflict of Interest:** None

Figure 1

fMRI findings in the pre-recovery phase (figure a-d). During verbal production, increased BOLD signal is observed in Broca's area and in the accessory para-median frontal motor area (figures 1a and b). In the verbal comprehension task, a stronger activation occurred in the left superior temporal gyrus and a bilateral activation in the planum temporal (figures 1c and d). Figures c and d show the BOLD activation superimposed on Diffusion Tensor Images with color coded diffusion directions [ blue: superior-inferior; red: left-right; green: anterior-posterior]. This allows the localization of the BOLD activation near to the arcuate fascicle (figure 1d) to be appreciated during the verbal comprehension task.

Table 1 : Language evaluation of MR during his period of mutism and after recovery using the Montréal Toulouse Protocol of the linguistic examination of aphasia [MT 86]; Evaluated domains are oral and written comprehension, oral and written production, rhythm and whistling. Cp = Comprehension; N = Normal; \* means impaired performance

<i>Test</i>	<i>During Mutism</i>	<i>After Language recovery</i>
Spontaneous interview	Mute*, (Cp =N)	N
Word /Nonword Repetition	0/15 *	15/15
Oral Cp (words + sentences)	56/57	57/57
Written Cp (words + sentences)	12/12	12/12
Oral Reading	O/33 * (Cp =N)	33/33
Spontaneous Writing	N	N
Written Naming	30/31	30/31
Rhythm recognition	4/5	5/5
Whistling	Possible, but poor melody*	*

Table 2: Memory evaluation of MR during his period of mutism and after recovery. Evaluated memory domains are: short-term memory, (digits span) learning and delayed recall/long term verbal memory (Rey Auditory-Verbal Learning test = RAVLT), immediate and delayed recall/long term visual (Rey Complex Figure = RCF), semantic knowledge, and autobiographical episodic and semantic memory. (Koppelman's AMI N = Normal; \* means impaired performance. During the period of mutism, all responses were given in writing.

<i>Test</i>	<i>During Mutism</i>	<i>After Language recovery</i>
Digit Span	4 (written)	5
RAVLT, learning	57/75	46/75
RAVLT delayed recall	12	14
RCF immediate recall	30/36	Not repeated
RCF delayed recall	30/36	Not repeated
Semantic knowledge	Focal loss (1996-2006)	Partial relearning
AMI childhood: A+S	21/21 +9/9	21/21 +9/9
AMI early adult life: A+S	21/21 +9/9	21/21 +9/9
AMI recent life 1996 -2006: A+S	0/21* +0/9*	9/21* +4/9*
AMI recent life 2006-2007: A+ s	21/21 +9/9	21/21 +9/9

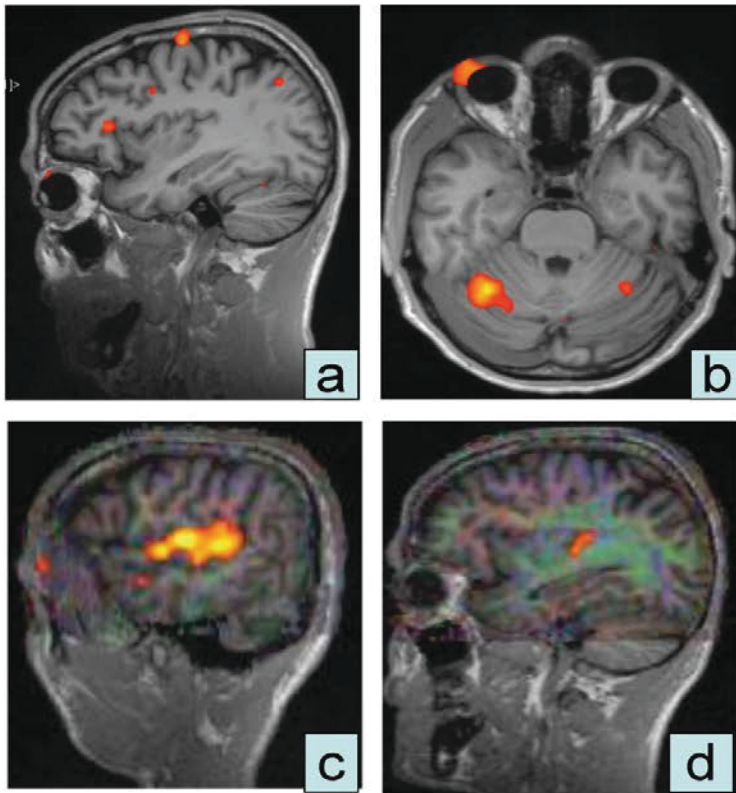


Fig 1: Brain activation during verbal production (a,b), during verbal comprehension (c ) and brain DTI (d)

References:

1. Maguire M, Seidi O, Baker M, Gupta A, Muwanga C. Acute mutism: a useful lesson. *Emerg Med J*. 2011; **28**(1): 82-3.
2. Kim YW, Shin JC, An YS. Treatment of chronic akinetic mutism with atomoxetine: subtraction analysis of brain f-18 fluorodeoxyglucose positron emission tomographic images before and after medication: a case report. *Clin Neuropharmacol*. 2010; **33**(4): 209-11.
3. Jacobsen T. Case study: is selective mutism a manifestation of dissociative identity disorder? *J Am Acad Child Adolesc Psychiatry*. 1995; **34**(7): 863-6.
4. Altshuler LL, Cummings JL, Mills MJ. Mutism: review, differential diagnosis, and report of 22 cases. *Am J Psychiatry*. 1986; **143**(11): 1409-14.
5. Turkstra LS, Bayles KA. Acquired mutism: physiopathy and assessment. *Arch Phys Med Rehabil*. 1992; **73**(2): 138-44.
6. Nagaratnam N, Fanella S, Gopinath S, Goodwin A. Prolonged abulia following putaminal hemorrhage. *J Stroke Cerebrovasc Dis*. 2001; **10**(2): 92-3.
7. Thorpe LU, Keegan DL, Veeman GA. Conversion mutism: case report and discussion. *Can J Psychiatry*. 1985; **30**(1): 71-3.
8. Sirois F. Hysterical mutism associated with a diagnosis of lung cancer. *Psychosomatics*. 2007; **48**(5): 452-3.
9. Kalman TP, Granet RB. The written interview in hysterical mutism. *Psychosomatics*. 1981; **22**(4): 362-3, 6.
10. Brand M, Eggers C, Reinhold N, Fujiwara E, Kessler J, Heiss WD, et al. Functional brain imaging in 14 patients with dissociative amnesia reveals right inferolateral prefrontal hypometabolism. *Psychiatry Res*. 2009; **174**(1): 32-9.
11. Ramasubbu R. Conversion sensory symptoms associated with parietal lobe infarct: case report, diagnostic issues and brain mechanisms. *J Psychiatry Neurosci*. 2002; **27**(2): 118-22.
12. Ersahin Y, Yazarbas U, Duman Y, Mutluer S. Single photon emission tomography following posterior fossa surgery in patients with and without mutism. *Childs Nerv Syst*. 2002; **18**(6-7): 318-25.
13. Ersahin Y. SPECT in cerebellar mutism. *Childs Nerv Syst*. 1998; **14**(11): 611-3.
14. Ersahin Y. Is splitting of the vermis responsible for cerebellar mutism? *Pediatr Neurosurg*. 1998; **28**(6): 328.
15. Kikuchi H, Fujii T, Abe N, Suzuki M, Takagi M, Mugikura S, et al. Memory repression: brain mechanisms underlying dissociative amnesia. *J Cogn Neurosci*. 2010; **22**(3): 602-13.
16. Kleinschmidt-DeMasters BK. Neuropathology of lightning-strike injuries. *Semin Neurol*. 1995; **15**(4): 323-8.
17. Cooper MA. Emergent care of lightning and electrical injuries. *Semin Neurol*. 1995; **15**(3): 268-78.
18. Freeman CB, Goyal M, Bourque PR. MR imaging findings in delayed reversible myelopathy from lightning strike. *AJNR Am J Neuroradiol*. 2004; **25**(5): 851-3.
19. Cherington M, Yarnell PR, London SF. Neurologic complications of lightning injuries. *West J Med*. 1995; **162**(5): 413-7.

20. Cherington M. Spectrum of neurologic complications of lightning injuries. *NeuroRehabilitation*. 2005; **20**(1): 3-8.
21. Wixted JT. On Common Ground: Jost's (1897) law of forgetting and Ribot's (1881) law of retrograde amnesia. *Psychol Rev*. 2004; **111**(4): 864-79.
22. Kopelman MD, Wilson BA, Baddeley AD. The autobiographical memory interview: a new assessment of autobiographical and personal semantic memory in amnesic patients. *J Clin Exp Neuropsychol*. 1989; **11**(5): 724-44.
23. O'Bryant SE, Gavett BE, McCaffrey RJ, O'Jile JR, Huerkamp JK, Smitherman TA, Humphreys JD. Clinical utility of Trial 1 of the Test of Memory Malingering (TOMM). *Appl Neuropsychol*. 2008; **15**(2): 113-6.
24. Maeder PP, Meuli RA, Adriani M, Bellmann A, Fornari E, Thiran JP, et al. Distinct pathways involved in sound recognition and localization: a human fMRI study. *Neuroimage*. 2001; **14**(4): 802-16.
25. Cronholm B, Lagergren A. Memory disturbances after electroconvulsive therapy. 3. An experimental study of retrograde amnesia after electroconvulsive shock. *Acta Psychiatr Scand*. 1959; **34**: 283-310.
26. Meeter M, Murre JM, Janssen SM, Birkenhager T, van den Broek WW. Retrograde amnesia after electroconvulsive therapy: A temporary effect? *J Affect Disord*. 2011.
27. Squire LR, Slater PC, Miller PL. Retrograde amnesia and bilateral electroconvulsive therapy. Long-term follow-up. *Arch Gen Psychiatry*. 1981; **38**(1): 89-95.
28. Calev A, Gaudino EA, Squires NK, Zervas IM, Fink M. ECT and non-memory cognition: a review. *Br J Clin Psychol*. 1995; **34** ( Pt 4): 505-15.
29. Akhtar S, Buckman J. The differential diagnosis of mutism: a review and a report of three unusual cases. *Dis Nerv Syst*. 1977; **38**(7): 558-63.
30. Christensen JA, Sherman RT, Balis GA, Wuamett JD. Delayed neurologic injury secondary to high-voltage current, with recovery. *J Trauma*. 1980; **20**(2): 166-8.
31. Ouellet J, Rouleau I, Labrecque R, Bernier G, Scherzer PB. Two routes to losing one's past life: a brain trauma, an emotional trauma. *Behav Neurol*. 2008; **20**(1-2): 27-38.
32. Cojan Y, Waber L, Carruzzo A, Vuilleumier P. Motor inhibition in hysterical conversion paralysis. *Neuroimage*. 2009; **47**(3): 1026-37.
33. Reinhold N, Markowitsch HJ. Emotion and consciousness in adolescent psychogenic amnesia. *J Neuropsychol*. 2007 Mar; **1**(Pt 1): 53-64.
34. de Lange FP, Toni I, Roelofs K. Altered connectivity between prefrontal and sensorimotor cortex in conversion paralysis. *Neuropsychologia*. 2010 May; **48**(6): 1782-8.

## SUPPLEMENTARY MATERIAL

### Appendix 1 : additional clinical data.

When MR returned home after the accident, he continued to suffer from amnesia for the previous ten year period. He did not remember anything about his daughters' childhood. He recognised his colleagues because he had been working for nearly 20 years in the same business, but did not recognise his professional activities between 1996 and 2006. He remembered Swiss politicians who were active until 1996, but not those who were in the Swiss parliament between 1996 and 2006. When the pictures of the terror attack of the 9/11 plane crash on the New York World Trade Centre were shown to him, he failed to identify the event. Instead, he suggested that these were taken from a movie. Similarly, when he was shown his own mobile phone, he could not identify it, as he had no memory of using that model of cell phone between 2000 and 2006. He remembered only those cell phones that he had used in 1996 for his work (GSM mobile phones) and was surprised to learn that such phones were no longer in use. He said that he had never heard of Tamagotchi games or DVD systems. He had no knowledge of I-pods, and was surprised to see very small electronic notebooks. Instead, he talked about videocassette recorders that were in standard use prior to 1996. His cardiovascular status was unremarkable during this period. He had no past or family history of psychiatric disorders and did not report any obvious secondary improvements.

Over the next three months his motor functions improved significantly, but the mutism and retrograde amnesia persisted. On one occasion, however, about six months after the accident, his wife did hear him utter two short but distinct sentences while awakening from a deep sleep, but the patient was too drowsy to have noticed it. He kept detailed records of all his written communications, from September 17<sup>th</sup>, 2006 onwards. He came to the clinics with his notebooks and would respond by writing or by nodding his head. After a few months he started to learn sign language and gradually



began to communicate by using his hands. However, he was independent to the extent that he did not require help in pursuing his daily routine activities. He had left his previous job as a sanitary installer, and had found new employment very rapidly as a machine driver in a recycling station, and had learnt sign language by attending a professional training school. He had asked for support from the disability insurance, which had been refused. He was able to help his children with their mathematics homework. After his initial injury, he learned about the events of previous decade with the help of his friends and relatives.

Regarding his behavior, his wife had noticed a change in his personality: he had become emotionally distant, was more forgetful and slower in decision making. These changes led to some difficulties in his interpersonal (e.g., with family) relationships.

## **Appendix 2 : Other experimental sessions**

*Diffusion Tensor Imaging (DTI)*: We performed a DTI tractography of the arcuate fasciculus bilaterally, using a streamline reconstruction of the fibre trajectories as supported by Trackvis software.<sup>33</sup> The seeding region of interest was placed on each side according to anatomical and radiological coordinates, and allowed to track the arcuate fascicles and to perform fractional anisotropy (Fa) and fibre density analysis on each side.<sup>33, 34</sup> The inter-hemispheric comparison, however, did not show any significant difference and Fa values were comparable to those obtained from healthy subjects' coordinates.<sup>33, 34</sup>

*Transcranial Magnetic Stimulation:*

**Method:** MR was seated in a comfortable reclining chair. A figure-of-eight shaped coil (7 cm inner diameter for each half) connected to a Bistim-module and two Magstim 200 magnetic stimulators (The Magstim Company, Dyfed, UK) was positioned on the scalp over the left M1. The hot spot for the right FPB muscle was defined as the lowest threshold site evoking a MEP response in FPB accompanied by a clear thumb flexion movement. The coil was positioned with the handle pointing backwards at an angle of 45° to the midline<sup>35</sup>. The hot spot was marked with a pen on the cap worn by the subject; this served as a visual reference against which the coil was positioned and kept in place by the experimenter. MEPs were recorded using surface EMG electrodes from the right flexor pollicis brevis (FPB) in bipolar belly-tendon arrangements (band-pass 10 Hz-2 kHz), using a Nicolet Viking electromyograph (Skovlunde, Denmark).

- **Resting motor threshold (rMT).** The resting motor threshold (rMT) was defined as the minimum TMS intensity (measured by altering the stimulator output intensity in one percent decrements) required to elicit at least five FPB MEPs > 50  $\mu$ V in ten consecutive trials. TMS stimulus intensities were then expressed as percentages of the right or left FPB rMT<sup>35-40</sup>.
- For MEP measurements, we averaged at least ten trials at 120% rMT for the FPB muscle of each side.
- **F – waves.** F-waves were evoked by a supramaximal stimulation of the median nerve at the wrist. The latencies of 18 F-waves were averaged for each side.
- **Central motor conduction time (CMCT).** Central motor conduction time was measured in the upper limbs, by calculating the difference between the latency of the MEPs to cortical stimulation and peripheral motor conduction time (PMCT) as assessed by using the F-wave latency:  $PCT (ms) = \frac{1}{2} (F + M - 1)$ , where F and M are respectively the onset latencies of F and M in milliseconds<sup>35-40</sup>.

**The findings from TMS** included MEP latencies of 23.51 ms  $\pm$  0.6 ms at the right and 23.03 ms  $\pm$  0.8 ms at the left FPB muscle. F-wave latency was 28.6 ms for the right median nerve, and 28.4 ms for the left median nerve, which is within the range of

normal values ( $26.6 \text{ ms} \pm 2.2\text{ms}$ ). M-latency was 4.8 ms after stimulation of the right and 4.6 ms after stimulation of the left median nerve. CMCT was 7.25 ms at the right side and 7.00 ms at the left side, given the fact that F-wave latency was symmetric. These values are normal ( $6.13 \pm 0.89$ )<sup>32</sup> and thus argue against any long-term consequences of the electrocution to the central motor system<sup>35-40</sup>.

Complete Table of Neuropsychological data

Domains Tested	Findings (Scores)	
	Pre-Recovery (October 2007)	Post Recovery (January 2009) (Day 10-30)
Handedness	Lateralization Index of Oldfield Epworth: 66.67%	- Identical
Mood Behaviour	Hamilton's Anxiety Depression (HAD) Scale: <b>Marginal 10/21</b>	HAD: N Anxiety:7/21, Depression:1/21
	<b>Emotional blunting</b>	<b>Hyperactivity</b>
Fatigue	Fatigue Assessment Instrument : <b>Normal</b>	<b>Increased with increase in physical and cognitive activity</b>

Language	Montréal Toulouse Protocol of the linguistic examination of the aphasia [MT 86]	Montréal Toulouse Protocol of the linguistic examination of the aphasia [MT 86]
	<ul style="list-style-type: none"> <li>• Interview: Good comprehension; mute, explained by writing, non-verbal gestures and signs</li> <li>• Words and non words repetition : impossible (0/15) <ul style="list-style-type: none"> <li>• Oral words Comprehension: normal (9/9)</li> <li>• Oral comprehension of sentences: within normal limits (35/38 with 2 auto corrections)</li> </ul> </li> <li>• Comprehension of written words: normal (6/6)</li> <li>• Written text Comprehension : normal (6/6)</li> <li>• Oral reading : impossible (0/33 ; no articulatory movements possible)</li> <li>• Spontaneous writing : fluent and normal</li> <li>• Writing of the letters: normal</li> <li>• Written naming: normal (30/31).</li> <li>• Dictation : normal (13/13)</li> <li>• Reading and repetition of numbers: impossible.</li> <li>• Calculation: normal.</li> <li>• Rhythm: 1 error in 5 trials (4/5).</li> <li>• Whistling a common French song “Au clair de la lune”: difficulty with the melody</li> </ul>	<ul style="list-style-type: none"> <li>• Interview: slightly aprosodic 7 day after recovery; normal 2 months after</li> <li>• Words and non words repetition : normal (15/15) <ul style="list-style-type: none"> <li>• Oral words Comprehension : normal (9/9)</li> <li>• Oral comprehension of sentences: 38/38</li> </ul> </li> <li>• Comprehension of written words: normal (6/6)</li> <li>• Written text Comprehension : normal (6/6)</li> <li>• Oral reading :normal</li> <li>• Spontaneous writing : fluent and normal</li> <li>• Writing of the letters: normal</li> <li>• Written Naming: normal (30/31).</li> <li>• Dictation : normal (13/13)</li> <li>• Reading and repetition of numbers: normal.</li> <li>• Calculation: normal.</li> <li>• Normal melody</li> </ul>

Praxis	<ul style="list-style-type: none"> <li>• Bucco-linguo-facial praxis: normal on verbal commands and imitation if no linguistic sound was asked but with hesitations whilst whistling</li> <li>• Naming the parts of the body: normal</li> <li>• Manipulation of the objects on verbal suggestions: normal</li> <li>• Rey's Complex Figure: normal (32/36)</li> </ul>	<ul style="list-style-type: none"> <li>• Normal even for linguistic sound, despite some hesitations</li> <li>• Gestural and constructive (cubes, watch) praxis: normal</li> </ul>
Spatio-temporal orientation	<ul style="list-style-type: none"> <li>• Normal</li> </ul>	<ul style="list-style-type: none"> <li>• Normal</li> </ul>
Memory	<ul style="list-style-type: none"> <li>• Digit span : impossible (mute) 4 if written</li> <li>• RAVLT: normal (written response) in learning, recognition (57 /75)</li> <li>• RAVLT : Delayed recall: lower limits (12)</li> <li>• Immediate recall of Rey's complex figure. normal (30/36)</li> <li>• Delayed recall : normal (29/36)</li> </ul>	<ul style="list-style-type: none"> <li>• Digit Span: 5</li> <li>• RAVLT learning : impaired (46/75)</li> <li>• RAVLT Delayed recall (14) and recognition: normal</li> </ul>
	<p><b>Autobiographical Memory:</b></p> <ul style="list-style-type: none"> <li>• AMI, Childhood, 21/21 + 9/9</li> <li>• AMI, Early Adult life 21/21+ 9/9</li> <li>• AMI, Recent life 1996/2006: 0/21+ 0/9</li> <li>• AMI, Recent life 2006/2007:21/21+9/9</li> </ul>	<p><b>Autobiographical Memory:</b></p> <ul style="list-style-type: none"> <li>• AMI, Childhood, 21/21 + 9/9</li> <li>• AMI, Early Adult life 21/21+ 9/9</li> <li>• AMI, Recent life 1996/2006: 9/21+ 4/9</li> <li>• AMI, Recent life 2006/2009:21/21+9/9</li> </ul>

Executive Functions	<ul style="list-style-type: none"> <li>• Luria Test: 3 perseverations; autocorrected.</li> <li>• Verbal fluency</li> <li>• Phonological fluency (12 in 1') : normal</li> <li>• Categorical fluency: normal.</li> </ul>	<ul style="list-style-type: none"> <li>• Luria gestural sequences: normal</li> <li>• Phonological fluency (10 in 1') : marginal</li> <li>• Stroop test : normal</li> <li>• Trail Making test : normal</li> </ul>
	<ul style="list-style-type: none"> <li>• Trail Making Test B: 53 seconds, normal.</li> </ul>	<ul style="list-style-type: none"> <li>• Trail Making Test B: normal</li> </ul>
	<ul style="list-style-type: none"> <li>• Pegboard Test: Left hand: 15 and 18, Right hand:14 and 13 (marginal for right hand)</li> </ul>	<ul style="list-style-type: none"> <li>• Pegboard Test: Both hands: 15 in 30 "</li> </ul>
Gnosis	<ul style="list-style-type: none"> <li>• Detection of body parts : 8/8</li> </ul>	<ul style="list-style-type: none"> <li>• Not repeated</li> </ul>
Gnosis	<ul style="list-style-type: none"> <li>• Columbia Test Score: 10/12 (normal)</li> <li>• Test of super imposed images: normal</li> </ul>	<ul style="list-style-type: none"> <li>• Test of super imposed images: normal</li> </ul>
	<ul style="list-style-type: none"> <li>• Dichotic Hearing</li> <li>- Laterality Index: -0.156 (<i>not suggestive of atypical language representation</i>)</li> </ul>	<ul style="list-style-type: none"> <li>• Dichotic Hearing not repeated</li> </ul>
Attention	<ul style="list-style-type: none"> <li>• Trail Making Test A: 34 seconds, normal.</li> </ul>	<ul style="list-style-type: none"> <li>• Trail Making Test A: 15<sup>th</sup> centile</li> </ul>